Chordal rupture
II: *Comparison between repair and replacement*

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SUMMARY During the period 1970–81, 183 patients underwent mitral valve surgery for chordal rupture. Of these, 82 (45%) patients were treated by mitral valve repair and 101 (55%) by mitral valve replacement. Mean age at surgery was 57 years. The early mortality was nine of 183 (4·9%) patients, of whom five had undergone replacement and four repair. During the follow up period (mean 3·6 years, range 0·8–12·2 years) a further 27 patients died; 23 of these had undergone mitral valve replacement and four mitral valve repair. Cerebrovascular events accounted for 35% of the deaths after mitral valve replacement and none of those after mitral valve repair. In 11 patients repair was technically unsatisfactory, and mitral valve replacement was undertaken at the same operation; a further five patients required late replacement (mean 1·4 years) for pronounced mitral regurgitation. Actuarial curves predict a six year survival of 68±5·7% (mean±SD) for all patients after mitral valve replacement compared with 88±6·9% (mean±SD) after repair (p<0·01). Actuarial survival curves favour mitral valve repair as the procedure choice for chordal rupture, and in isolated posterior cusp repair breakdown of the repair is a rare occurrence.

With the introduction of open heart surgery in the 1950s effective surgical treatment for mitral regurgitation became available for the first time.[1-3] In many cases of mitral regurgitation secondary to ruptured chordae tendineae there is no shortage of relatively normal valve tissue. This fact, together with the known problems associated with prosthetic valves, has stimulated the development of conservative procedures in this condition.

McGoon[4] in 1958 was the first to use a specific technique for the repair of ruptured chordae although before this the condition had been treated with annuloplasty.[5 6] Since then there has been a substantial number of reports dealing with various repair techniques and the results obtained with them.[7-28] In view of the initially rather variable outcome following repair others have preferred mitral valve replacement.[29-32]

Recently, the advantages of repair over replacement in selected cases of mitral regurgitation have become clear, but there are no large series comparing the two operations in cases due solely to ruptured chordae. In

this paper we have reviewed all cases of surgically treated ruptured chordae seen recently at this hospital and made such a comparison.

Patients and methods

The case records of all 213 patients found to have ruptured chordae at open heart surgery for mitral regurgitation in the years 1970–81 inclusive were reviewed. Patients with non-rheumatic mitral regurgitation due to floppy valves caused by a dilated annulus (without ruptured chordae) were excluded from the study. In patients in whom there had been extensive destruction or retraction of valve tissue secondary to previous rheumatic heart disease, surgery, or bacterial endocarditis repair was clearly not feasible, and these patients were also excluded. No patients who presented with ruptured papillary muscles have been included (one during this period) nor have patients who underwent additional surgery (for example, aortic valve replacement (16), tricuspid plication (9), and coronary artery bypass grafting (5)). The remaining 183 patients represent a population that might reasonably have been treated with mitral repair or replacement and form the basis of this review. The mean age at the time of surgery was 57 (range 4–78) years; 87

Total patients
(n=183)
/   \
Attempted repair   Elective replacement
(n=82)   (n=101)
/   \
Successful repair   Failed repair/mitral valve replacement
(n=71)   (n=11)
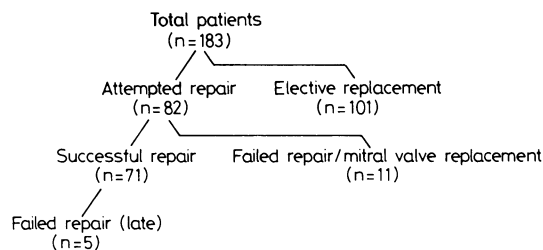/
Failed repair (late)
(n=5)

Fig. 1 *Mitral valve surgery for chordal rupture.*

were men and 96 women (ratio of 1 : 1·1). Of the 183 patients, 82 (45%) underwent mitral valve repair and 101 (55%) mitral valve replacement (Fig. 1).

MITRAL VALVE REPAIR
Of the eighty-two patients who underwent mitral valve repair (mean ($\pm$SD) age 59$\pm$12 years) no clear aetiology for the chordal rupture could be defined in 75 (91%), and these patients are subsequently referred to as having spontaneous rupture. In seven (9%) documented bacterial endocarditis had occurred on a previously normal valve. Fifty-nine (72%) patients were in sinus rhythm, twenty-two (27%) in atrial fibrillation, and one in complete atrioventricular block. Seventy (85%) patients had rupture of the posterior chordae, ten (12%) rupture of the anterior chordae, and two rupture of the chordae to both mitral valve cusps. It is not the policy at this hospital routinely to catheterise all patients with valvular heart disease.[33] In this series 30 (37%) patients in the repair group underwent catheterisation. Preoperatively, 51% of patients undergoing mitral repair were classified (New York Heart Association) as being in functional class III or IV.

MITRAL VALVE REPLACEMENT
Of 101 patients who underwent mitral valve replacement (mean ($\pm$SD) age 56$\pm$15 years) chordal rupture was spontaneous in 79 (78%). Sixteen (16%) had documented bacterial endocarditis, 11 (11%) on a normal valve and five (5%) on a valve previously affected by rheumatic carditis. Five (5%) had ischaemic heart disease and one (1%) ruptured chordae complicating acute rheumatic fever. Fifty-eight (57%) were in sinus rhythm and the remaining 43 (43%) in atrial fibrillation. Ruptured chordae affected the posterior cusp in 31 (31%), the anterior cusp in 58 (57%), and both mitral cusps in 12 (12%) patients; 47 (47%) patients in this group underwent cardiac catheterisation. Preoperatively, 56% were in functional class III or IV (New York Heart Association).

SURGICAL TECHNIQUE
In all cases the heart was exposed via a median ster-

notomy incision. Cardiopulmonary bypass was established with cannulation of the ascending aorta and both cavae. Before 1978 the operation was performed under moderate hypothermia (32°C) with the heart fibrillating and intermittent periods of aortic cross clamping. Thereafter, the technique of cold cardioplegic arrest has been used with systemic cooling to 28°C. The mitral valve was exposed by a vertical incision in the left atrium and the diagnosis of ruptured chordae confirmed by direct inspection. Eighty two patients initially underwent mitral valve repair based on the method of Carpentier *et al*.[25] In cases of ruptured chordae to the posterior leaflet (85% of patients) a trapezoid segment of the leaflet incorporating the ruptured chordae was excised, and the leaflet repaired with interrupted sutures of 4-0 Ethiflex. In addition, one circumferential 2-0 Ethiflex suture was used to narrow and reinforce the annulus. Where chordae to the anterior leaflet had ruptured a triangular segment of that cusp was excised. If both cusps were involved then segments of both leaflets were excised as appropriate. The repair technique has evolved during the time course of the series and descriptions of the earlier,[21] and more recent,[22 26 28] methods have been published. The competence of the mitral valve was then tested by rendering the aortic valve incompetent and allowing the left ventricle to fill with blood.[34] In 11 patients mitral valve repair was technically unsatisfactory, and mitral valve replacement was carried out at the same operation; this subgroup included seven patients with ruptured chordae to the posterior leaflet, two with ruptured chordae to the anterior leaflet, and the two in whom both leaflets were affected.

Other surgeons favoured mitral valve replacement as the procedure of choice. The choice of valve was left to the individual surgeon. Forty six (46%) patients had a Starr-Edwards 6120 prosthesis, 27 (27%) a Björk-Shiley tilting disc valve, 18 (18%) a Carpentier-Edwards porcine xenograft, nine (9%) an aortic homograft, and one an Omniscience tilting disc valve. The prostheses were inserted with interrupted over and over 2-0 Ethiflex sutures. The left atrial appendage was not excised in either group of patients. After surgery all patients with mechanical prostheses underwent anticoagulation with warfarin to maintain the British Standard prothrombin ratio at 2·5–3·0. In addition, patients with a biological valve in atrial fibrillation and those who had undergone mitral valve repair and remained in atrial fibrillation underwent formal anticoagulation.

Results

EARLY MORTALITY
After mitral valve repair the early mortality rate in hospital was four of 82 (4·9%) patients. Three of these

patients had undergone simple mitral repair. One died of low output failure, one of haemorrhage, and one of an acute myocardial infarct. One further patient died of valve dehiscence caused by prosthetic endocarditis after attempted mitral repair followed by valve replacement. In the valve replacement group the hospital mortality rate was five of 101 (5%). Four patients died of low output failure and one of prosthetic endocarditis (Table 1).

LATE MORTALITY

For the series the mean follow up period was 3·6 years (range 0·08–12·2 years). Eleven (6%) patients were lost to follow up as they returned to their country of origin; one patient was in the repair group and 10 were in the replacement group. During this time a further four patients died in the mitral repair group: two of low output failure, one of bacterial endocarditis, and one suddenly of unknown cause. In the replacement group a further 23 patients died. Table 1 shows the causes of death. In one patient death was due to pulmonary oedema caused by thrombosis of a Björk-Shiley valve as a result of stopping oral anticoagulant treatment. Actuarial survival curves[35] for all patients predict a six year survival of 88±6·9% (mean±SD) for those patients who underwent mitral valve repair compared with a six year survival of 68±5·7% for those who underwent mitral valve replacement (p<0·01) (Fig. 2a). Patients who had attempted repair followed by mitral valve replacement have been included in the valve replacement group for the purposes of actuarial analysis. Actuarial survival at six years was not influenced by cardiac catheterisation (catheterised group 80±6·1%; non-catheterised group 80±7·7% (mean±SD) not significant (Fig. 2b).

Table 1 *Early and late mortality after mitral valve surgery. (Late mortality replacement group includes patients with failed repairs/replacements.) (Figures are numbers of patients)*

Causes	Repair	Replacement
Early mortality		
Low output failure	1	4
Haemorrhage	1	
Acute myocardial infarct	1	
Subacute bacterial endocarditis	1*	1
Overall (%)	4/82 (4·9)	5/101 (5)
Late mortality		
Cerebrovascular events	0	8
Low output failure	2	6
Sepsis	0	3
Subacute bacterial endocarditis	1	2
Sudden death	1	1
Hepatic failure	0	1
Haemorrhage	0	1
Prosthetic thrombosis†	0	1
Overall (%)	4/68 (6)	23/106 (22)

*Prosthetic endocarditis after failed repair.
†Patient stopped oral anticoagulant treatment.

Table 2 *Causes of death in eight patients suffering cerebrovascular accidents after mitral valve replacement*

Patient's age (sex)	Valve	Rhythm	Pathology
71 (M)	SE	Sinus rhythm	Embolism
67 (F)	SE	Atrial fibrillation	Haemorrhage
61 (F)	SE	Sinus rhythm	Embolism
40 (F)	SE	Sinus rhythm	Embolism
47 (M)	BS	Sinus rhythm	?
52 (M)	BS	Atrial fibrillation	Embolism (2)
65 (M)	BS	Sinus rhythm	Haemorrhage
40 (M)	Aortic homograph	Sinus rhythm	Embolism (2)

SE, Starr-Edwards; BS; Björk-Shiley.

VALVE DYSFUNCTION AFTER REPAIR

Five patients required late mitral valve replacement (mean 1·4 years after repair) for pronounced mitral regurgitation; four had anterior chordal rupture and one posterior chordal rupture. There was no additional mortality as a result of reoperation.

THROMBOEMBOLISM

A thromboembolic event was defined as a sudden new postoperative neurological disturbance resulting in a permanent deficit. There were no thromboembolic events affecting other major arteries in patients in this series. In the repair group one patient had a non-fatal cerebrovascular event, but thromboembolism was not a cause of death in this group. In the replacement group thromboembolic events resulting in a permanent neurological deficit occurred in six patients: three with a Björk-Shiley valve, two with a Starr-Edwards 6120 valve, and one with an aortic homograft. Fatal cerebrovascular events were the cause of death in eight of 23 (35%) patients in the valve replacement group (Table 2) (mean age 55 (range 40–71) years); of the eight, five were men and six were in sinus rhythm. Five patients sustained cerebral emboli, and two of these had had a previous embolism before the event that caused death. In two the cause of death was cerebral haemorrhage. In the remaining patient the cause of the cerebrovascular event was unknown. Confirmation at necropsy was available in five patients. The thromboembolic rate for the mitral valve repair group was, therefore, 0·2/100 patient years compared with that for the replacement group of 1·4/100 patient years.

FUNCTIONAL CLASS

Postoperatively, 82% of the replacement group and 83% of the repair group were in functional class I (New York Heart Association classification).

Discussion

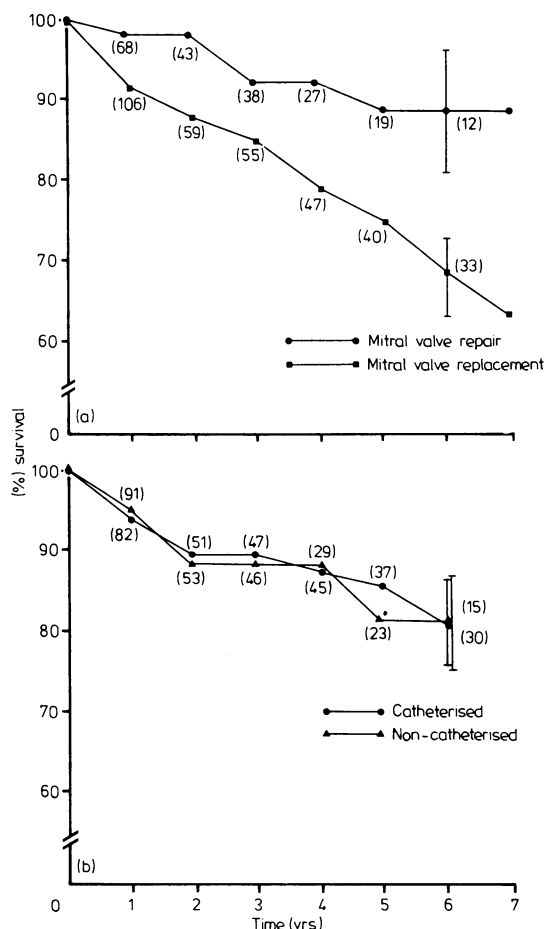Not all cases of ruptured chordae will require surgery.

Fig. 2 *Mean (±SD) percentage survival for all patients after mitral valve surgery: (a) repair v replacement groups and (b) catheterised v non-catheterised groups.*

In the eight cases reported by Ronan *et al.*[36] three improved and were maintained on medical treatment six, 12, and 36 months after their initial symptoms. These cases represent the minority, however, and for most the choice lies between mitral valve replacement and repair.

The decision on when to operate in cases of mitral regurgitation may not be straightforward. Kay *et al.*[23] stated that the criteria should be solely symptomatic, but a recent study[37] of prognostic factors in surgery for non-rheumatic mitral regurgitation found that variables such as short history (<1 year) and a normal left ventricular end-diastolic volume index (<100 ml/m^2) were favourable prognostic factors. An ejection fraction of <0.5 was uniformly associated with a poor outcome. These results suggest that operative inter-

vention should be considered earlier rather than later before irreversible myocardial damage occurs.

Having decided to operate the choice of surgery lies between repair and replacement. This decision is best made after inspection of the valve, and Carpentier *et al.*[25] have published detailed indications for and against repair depending on the precise cause of the regurgitation. Other factors to be considered include the experience of the surgeon in the repair technique and the results of previously published series. The results of this present series are not strictly comparable with those of earlier reports as there are no other large series dealing exclusively with cases of mitral regurgitation secondary to ruptured chordae.

Comparison between the repair and replacement groups is meaningful only if there are no significant differences in the incidence of risk factors between the two groups. The two groups were comparable for age and preoperative functional status. In the repair group, however, 85% of patients had rupture of the posterior cusp whereas in the replacement group the predominant lesion was anterior chordal rupture (57%). In the early part of the series one of us (MP) electively treated all cases of ruptured chordae by mitral valve repair if technically feasible. It became clear during the series that the leaflet affected by chordal rupture influenced the success or otherwise of the repair. Thus anterior leaflet chordal rupture was associated with a disproportionately high incidence of intraoperative and late failure of repair. Of the 10 patients with anterior rupture who underwent repair, two required mitral valve replacement at the initial operation and four required reoperation for pronounced mitral regurgitation. Furthermore, the two patients in the repair group who had ruptured chordae to both the anterior and the posterior cusps required immediate mitral replacement because of a technically unsatisfactory repair. This conforms with the experience of Carpentier *et al.*[25] who found that if resection of one fourth or more of the anterior leaflet was required to control regurgitation, then repair was contraindicated. Similarly, Gerbode *et al.*[19] treated four cases of chordal rupture to the anterior leaflet and two out of the three with chordal rupture to both leaflets in their series with mitral valve replacement. Other authors, although not commenting on the actual incidence of anterior leaflet ruptured chordae, do not seem to have had an excess of failed repairs in this group.[23] [26] It is now our policy to attempt mitral valve repair only on patients in whom there is isolated posterior chordal rupture.

The early mortality for mitral repair or replacement was very similar at 4.9% and 5.0% respectively, which compares favourably with other series for repair[23-25] at 4.2–6.0% and for replacement[38-42] at 6.0–13%. The differences in late mortality between the two

groups are striking. There were no fatal cerebrovascular episodes in the repair group, but there were eight deaths in the replacement group. These episodes do not appear to be related to atrial fibrillation as the incidence of atrial fibrillation was low in this subgroup (2/8) despite the higher incidence of atrial fibrillation in the replacement group as a whole (43% v 27%). Of eight patients, death was due to thromboembolism, a complication of the prosthesis, in five, to cerebral haemorrhage complicating anticoagulant therapy in two, and the cause of the cerebrovascular event was unknown in one. The results in the replacement group were not biased by one particular prosthesis performing badly (Table 2). The incidence of non-fatal thromboembolism in both the repair and replacement groups was low when compared with other series.[23 38 42 43] This may be related in part to the fact that 10 of the 11 patients lost to follow up were in the valve replacement group. Difficulties arise when defining a non-fatal cerebrovascular event, and much of the variation in the reported incidence is related to differences in definition. The Carpentier-Edwards prosthesis was implanted only late in the series; therefore the absence of thromboembolic events with this valve may be related to the short follow up in this subgroup.

Other complications—for example, bacterial endocarditis and haemolytic anaemia—are also more frequent after valve replacement, although the numbers in this series are too small to analyse. Haemolytic anaemia is not a unique complication of prosthetic valves as there is at least one reported case of haemolysis occurring during the breakdown of a repair.[44]

The results of this series are, therefore, in agreement with those of the series of Yacoub *et al.*[27] which is the only other sizeable series comparing the two operations in a relatively uniform population, in demonstrating the significantly greater survival of repair over replacement. In the survivors both operations produced a similar improvement in functional status. In Yacoub's series, however, only a proportion of patients had mitral regurgitation secondary to chordal rupture; furthermore, mitral repair was compared with mitral replacement using a free aortic homograft in all patients and not a mechanical prosthesis.

The surgical management of valvular heart disease without previous cardiac catheterisation is controversial.[33 45-49] Our present policy is to perform preoperative catheterisation only on patients in whom doubt remains concerning the severity of the valve lesion after full non-invasive investigation—including M-mode and cross sectional echocardiography—or when symptoms suggesting ischaemic heart disease form part of the clinical history. Although occult coronary artery disease may be missed in patients undergoing valve surgery without previous catheterisation, no patient in either group developed angina in the follow up period nor did undetected coronary artery disease in the non-catheterised group cause a reduction in the predicted six year actuarial survival, which was identical in both the catheterised and non-catheterised groups (80%). This was despite the reduced preoperative exercise tolerance in the non-catheterised group (64%, functional classes III and IV) compared with the catheterised group (45%, functional classes III and IV). Our experience suggests that cross sectional echocardiography is the most useful investigation in patients with chordal rupture. The size of the mitral annulus can be assessed accurately, and flail mitral leaflets when present are readily apparent prolapsing into the left atrium during ventricular systole. On occasions a curled up mitral leaflet from partial chordal rupture may appear to be indistinguishable from a vegetation in bacterial endocarditis. Attenuated mitral chordae can result in an echocardiographic appearance indistinguishable from chordal rupture; however, both pathological processes result in severe mitral regurgitation, and the surgical treatment is therefore similar.

In conclusion, mitral valve surgery for chordal rupture has an acceptably low early mortality. Actuarial survival curves favour mitral valve repair as the surgical treatment of choice in the treatment of posterior chordal rupture, mainly because of the lack of thromboembolic events after repair. Since repair of the anterior leaflet is prone to dehiscence mitral repair should be reserved for patients with isolated posterior chordal rupture.

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